EPICARDIAL ACTIVATION DURING ENTRAINMENT AND TERMINATION OF SINGLE LOOP REENTRY IN VIVO

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The epicardial activation sequence during entrainment and termination of single reentrant loops in a syncytium without anatomically predetermined pathways has never been shown. For in situ mapping of the total atrial surface of the canine heart a special electrode with 127 bipolar electrodes in a flexible nylon matrix was designed. After placement of the electrode, sustained atrial flutter (AF) was induced in 4 dogs with sterile pericarditis. Epicardial maps showed a single wavefront circulating around an arc of functional conduction block in the proximity of the atrio-ventricular ring or around a combined obstacle, with the arc being contiguous with one of the caval veins. Atrial pacing at a cycle-length (CL) 5-25 msec shorter than the spontaneous CL could result in an antidromic (opposite to the spontaneous wavefront) stimulated wave that collided with the orthodromic wavefront of the previous beat at a constant site. The site of collision shifted with changes in the CL of stimulation. The functional arc of block did not change in length or configuration. At a critical CL, the orthodromic wavefront arrived at the slow conducting zone of the circuit before refractoriness expired, resulting in conduction block. During the following stimulated cycle, electrode sites distal to this block were activated from a different direction and at a shorter coupling interval. The occurrence of conduction block was determined by the CL of stimulation and the number of stimulated beats, which was usually ≥ 4. A longer train could re-induce the same or a different, potentially shorter reentrant circuit during stimulated cycles following the beat that terminated reentry. Thus, for a given CL of stimulation there was an "optimal" number of stimulated beats for successful termination of the arrhythmia.
Conclusions: Epicardial activation maps confirm the suggested mechanism for entrainment of AF. While the activation sequence during termination of single loop reentry is similar to the sequence seen during termination of ventricular tachycardia, the required number of stimulated beats might indicate differences in excitability within the reentrant pathway.

MULTIPLEXING STUDIES DURING ENTRAINMENT AND INTERRUPTION OF ATRIAL FLUTTER TO CHARACTERIZE DOUBLE POTENTIALS

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The center of the atrial flutter (AFl) reentry circuit in our sterile pericarditis canine model is characterized by double potentials (DP), defined as 2 discrete deflections per beat separated by an isoelectric interval. To test the hypothesis that these DP reflect functional conduction block rather than slow conduction, we studied 10 episodes of AFl in 6 dogs during transient entrainment and interruption of the AFl while pacing from the inferior left atrium or the right atrial appendage. A multiplexing system was used to record simultaneously from 95 bipolar electrodes on the right atrium (site of reentry). During AFl, there was more than one area of slow conduction in the reentry circuit. During RP, the interval from the stimulus artifact to the 1st potential of the DP remained constant, but the interval to the 2nd potential of the DP prolonged, and the morphology of the DP remained unchanged. Interruption of AFl occurred when the orthodromic wave front from the pacing impulse blocked in an area of slow conduction in the reentry circuit. When the 2nd potential of the DP was orthodromically distal to this area of block, interruption of the AFl was associated with disappearance of this potential. When the 2nd of the DP was proximal to the area of block, there was no localized conduction block to the 2nd DP. The response of the 2nd potential of the DP at the time of interruption of AFl strongly supports the conclusion that DP do not represent slow conduction through the center of the reentry circuit, but rather represent functional conduction block, with each potential of the DP reflecting activation on either side of the area of functional block.

Epicardial Mapping of Ventricular Tachycardia Induced by a Large Premature Stimulus Over Nontransmural Infarcts

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Mapping has recently shown that electrically induced ventricular tachycardia (VT) and ventricular fibrillation (VF) both begin as figure-8 reentry: VT with a longer cycle length from spared tissue adjacent to an infarct (inf) and VF with a shorter cycle length from tissue around the electrode from which a large premature S2 is given. These results suggest the cycle length of the figure-8 rather than the mode of induction determines the type of arrhythmia. Thus a protocol similar to that by which a VF threshold is determined may induce VT rather than VF when performed in the spared tissue over an inf. We tested this hypothesis in 10 dogs in which the proximal LAD was partially occluded for 30 min, totally occluded for 90 min and then reperfused. Four days later a 3x3 cm plaque containing 121 recording electrodes was centered over the epicardial inf area. Ten S1 stimuli were delivered from a total of 33 RV and LV sites outside the inf. An S2 was given to the epicardium over the inf from the center of the recording plaque. S2 was given decrementally to scan diastole and increased in 10 mA steps until VT or VF was induced. Sustained VT was induced repeatedly from 23 and VF from 10 S1 sites ($p<0.04$, chi-sq). The S2 was 39 ± 16 mA for VT and 56 ± 13 mA for VF ($p=0.004$, t-test). The mean difference in the arrhythmia cycle lengths for the initial 6 cycles was 153 ± 33 ms for VT and 110 ± 8 ms for VF ($p<0.001$, t-test). Mean transmural inf extent was 80% in 5 dogs with VT from all S1 sites, 60% in 3 dogs with VT from some and VF from other S1 sites, and 20% from 2 dogs with VF from all S1 sites. VT isochronal maps showed figure-8 reentry with the slow central part of the pathway oriented towards the region of earliest S1 activation. Thus, a large S2 over a nontransmural inf induces VT if the spared myocardium is thin. Besides verifying the hypothesis, this study introduces a useful animal model of sustained monomorphic VT in which the location (the S2 region) and direction (towards the region of the early S1 activation) of the figure 8 pathway is known a priori and in which different morphologies of sustained VT can be produced by changing the S1 site.

Tuesday, March 20, 1990
4:00PM–5:30PM, Room 43
Programmed Stimulation: Prognostic Observations

LONG-TERM OUTCOME OF ELECTROPHYSIOLOGICALLY-GUIDED THERAPY FOR HEMODYNAMICALLY-TOLERATED SUSTAINED VENTRICULAR TACHYCARDIA IN CORONARY ARTERY DISEASE.

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Among patients with sustained ventricular tachyarrhythmias, only cardiac arrest survivors have defined actuarial outcomes using electrophysiologically- (EP) guided therapy (cumulative 4-yr sudden death rate of 20% for EP suppressed and 70% for non-suppressed). We therefore analyzed survival in 75 coronary artery disease patients presenting with hemodynamically-tolerated monomorphic sustained ventricular tachycardia. Mean age was 63 years, with mean ejection fraction of 34%. 88% of patients initially had inducible sustained ventricular tachyarrhythmias during the drug-free EP study; 15% were ultimately suppressed by antiarrhythmic agents, and 13% by surgery. The non-suppressed group received amiodarone (56%), conventional agents (21%), sotalol (12%), surgery (2%), or automatic defibrillator (AICD) (9%). During a mean follow-up of 34±26 mos, there was 0% sudden death in the suppressed group vs a cumulative sudden death rate, at 1 and 4 years, respectively, of 7% and 13% for the non-suppressed group.
Conclusions: Long-term survival of coronary artery disease patients treated for hemodynamically-tolerated sustained ventricular tachycardia is better than that established for cardiac arrest survivors. Hence, uniformity of clinical presentation, as well as EP inducibility status, are needed to properly compare therapeutic outcomes in patients with "malignant" ventricular tachyarrhythmias.